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1: Curr Opin Investig Drugs. 2001 Jul;2(7):959-66.

Immunization against tumor cell surface complement-regulatory proteins.

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Complement is an enzymatic cascade that results in the release of pro-inflammatory anaphylatoxins, C3b deposition and the assembly of the membrane attack complex (MAC), which results in cell lysis. Cells express complement regulatory proteins or inhibitors to protect themselves from bystander attack by complement. Expression of the complement-regulatory proteins CD55, CD46 and CD59 are deregulated in cancer with tumors showing loss of one or more inhibitors and strong overexpression of others. This results in tumors that are resistant to attack by complement and is a major limitation in the use of monoclonal antibodies as monotherapies. However, tumor sensitivity to complement can be restored by co-administration of antibodies that bind to the functional domains of complement-regulatory proteins. Overexpression of complement-regulatory proteins on tumors also makes them potential targets for cancer vaccines. However, these vaccines have to be carefully designed to induce immune responses that recognize inhibitors overexpressed on tumors and that do not detect the levels expressed by normal cells. A human anti-idiotypic antibody that mimics CD55 has been used successfully in over 200 colorectal cancer and osteosarcoma patients. 70% Of patients show CD55-specific immune responses with no associated toxicity. Similar vaccines targeting CD46 and CD59 would eliminate any cell overexpressing a complement inhibitor. Any remaining tumor cell or any tumor cell that loses complement-regulatory proteins in response to therapy would become highly susceptible to in situ complement deposition. In summary, targeting complement-regulatory proteins is a very attractive approach to tumor therapy, although great care must be taken in preventing normal tissue recognition as this could lead to

uncontrolled complement deposition and massive cell lysis.

Publication Types:
- Review
- Review, Tutorial

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